Current Practice

PRACTICAL NEUROLOGY

Spinal Cord Compression—II

BERNARD HARRIES,* M.B., F.R.C.S.

British Medical Journal, 1970, 1, 673–676

(Last week Mr. Harries discussed the types of paraplegia and compression due to trauma and tumours. The article concludes this week.)

Outlines of Causation

Infections

There are three localized infections which may give rise to paraplegia.

Tuberculosis caries of the spine (Pott's paraplegia)—The cord compression is usually due to the presence of tuberculous granulation tissue within the spinal canal. Vertebral collapse (often involving two adjacent vertebral bodies and the intervertebral disc) is usually present, though the wedging of the vertebrae is not thought to be the prime cause of the paraplegia. A paravertebral abscess is also often present, and because it is in continuity with the spinal canal through an intervertebral foramen may, if tense, contribute to the cord compression. Occasionally bony or cartilaginous sequestra may also compress the cord. Pott's paraplegia is generally slow in onset and is associated with progressive activity of the disease when this has been untreated or inadequately treated. It usually arises during the early weeks or months of the disorder. Paraplegia of sudden onset in the presence of tuberculous caries is generally due to infarction of the cord.

Treatment of spinal caries entails immobilization, chemotherapy, and usually relatively early operative removal of pus, caseous material, and infected bone accompanied by bone-chip grafting to encourage stable union. The operation is by an anterolateral approach. Laminectomy is avoided.

Chronic staphylococcal osteomyelitis is an occasional cause of cord compression. In many ways it resembles tuberculous osteitis. It is insidious in onset, and the patient may have none of the stigmata of pyococcal infection. Though the radiological appearances of the affected vertebrae may be suggestive the distinction between staphylococcal osteitis and tuberculous caries may be confirmed only at operation.

Acute pyococcal (staphylococcal) extradural abscess may cause a paraplegia of overwhelming suddenness and is accompanied by much pain in the back, pyrexia, and leucocytosis. The infection reaches the spinal canal by the blood stream from some often unremarkable cutaneous infection. Very urgent decompression to evacuate the pus and appropriate antibiotic therapy is essential if permanent paraplegia from cord infarction is to be avoided

* Consultant Neurosurgeon, University College Hospital; Honorary Consultant Neurosurgeon to the Whittington Hospital.

In infants and children *infected lumbar dermoid cysts* may also give rise to an acute or subacute extradural abscess and a cauda equina paralysis of rapid onset. The association is so common that the investigation and surgical exploration of any small midline sinus in the lumbo-sacral region is to be advocated in infants.

Degenerative Disorders

Spontaneous subluxation and luxation of the cervical spine as a consequence of rheumatoid arthritis and steroid therapy are becoming a rather common cause of tetraparesis. Disruption of the cruciate ligaments of the odontoid process can result in a slowly progressive atlanto-axial dislocation. Softening and stretching of the ligaments of the intervertebral joints causing dislocation in the mid-cervical region is also not infrequent after prolonged steroid therapy.

Fortunately the paresis, which involves the upper and lower limbs and sometimes the bladder, is usually rather slow and insidious in onset. The sensory symptoms and wasting which occur in the hands may be misinterpreted or confused with the primary rheumatoid changes. Treatment by immediate skull traction and subsequent spinal fusion offers a very reasonable outlook and usually useful relief of the paretic symptoms.

Cervical spondylosis, some degree of which is almost ubiquitous in the middle aged and elderly, is sometimes associated with paraparetic or tetraparetic symptoms. Males over 50 are usually affected. The precise relationship between the two conditions is not clearly understood, but it is generally believed that as the canal becomes narrowed by the progressive degeneration protrusion, and sclerosis of one or more cervical discs, the spinal cord suffers increasing attrition and flattening of its anterior aspect. Secondary ischaemic changes in the territory of the anterior spinal artery produce widespread but patchy demyelination distal to the lesion. Clinically stiffness, weakness, and incoordination of the lower limbs is accompanied by sensory impairment in the fingers and hands. This may progress gradually to a florid spastic tetraplegia in which the lower limbs are more affected than the upper and in which motor changes predominate.

Surgical treatment of the condition remains far from satisfactory, but attempts at decompression by cervical laminectomy are currently being replaced by an anterior approach in which the sclerotic disc is removed and replaced by a "dowel" bone graft taken from the iliac crest. The results of this procedure remain to be evaluated but are promising. Careful preliminary assessment, which must include plain *x*-rays and myelography to determine which disc space or spaces are mainly affected, is essential.

Disc herniation in the thoracic region is a rare cause of paraplegia of sudden onset usually accompanied by much midline back pain and root pains. The sudden compression of the cord may result in severe though usually incomplete paraplegic symptoms. The prognosis with or without surgical treatment is poor.

Disc herniation in the lumbar region usually results in the familiar lumbar backache and sciatica when the protruding fragment impinges on one of the lower lumbar nerve roots as it emerges from the dura mater. With this condition we are not concerned here.

Occasionally, however, a large more centrally placed protrusion may rupture and "sequestrate" into the spinal canal and cause an abrupt cauda equina paralysis of greater or lesser severity. Numbness of the perineum and bladder symptoms such as retention or incontinence are usually prominent features. This condition is one of real urgency. Only prompt localization of the lesion by myelography and removal of the dislodged fragment surgically offers a good chance of recovery of bladder function and relief of a disabling cauda equina paresis. At the onset pain in the back and legs is a marked feature of this condition, but the pain tends to abate rather rapidly, thus masking the need for immediate surgical attention without which the results of treatment are poor.

Senile osteoporosis, Paget's disease, and osteoporosis due to steroid therapy are all occasional causes of paraparesis or paraplegia.

Differential Diagnosis

It is possible here to refer only to the commoner disorders which may cause difficulty or doubt when considering a diagnosis of cord compression. In the main the history of the illness and the physical signs if carefully elicited will exclude the likelihood or otherwise of compression. In most cases examination of the lumbar cerebrospinal fluid, including manometry, and if doubt still exists, myelography, will confirm or disprove the presence of a "block" in the spinal subarachnoid space.

Among the conditions likely to give rise to confusion are multiple sclerosis, "transverse myelitis," the variants of meningo-encephalo-myelitis, Landry's ascending paralysis (due to whatever cause), and the acute infective polyneuritides of which the Guillain-Barré syndrome is perhaps the commonest. Tabes dorsalis is unlikely to give rise to clinical difficulty, and confirmation will come from the serology. Very rarely a gummatous infiltration within the spinal canal may mimic a slowly growing tumour and emphasizes the importance of a routine W.R. examination of blood and cerebrospinal fluid in all cases of slow cord compression. Operative removal of a gumma may result in irrecoverable ischaemic changes in the cord. Subacute combined degeneration must be considered in the differential diagnosis of progressive weakness and sensory impairment of the limbs and sometimes the condition occurs before the full picture of pernicious anaemia is manifest.

Of the more abrupt causes of paraplegia "anterior spinal artery thrombosis," with the characteristic sparing of the posterior columns, is perhaps the commonest. It is sometimes associated with some other underlying disorder such as a blood dyscrasia, carcinomatosis, or arteriopathy. A rather less definite and less understood cause of paraplegic symptoms of slow onset is the so-called "arteriopathic myelopathy." Evidence of arterial disease elsewhere and normal cerebrospinal fluid and myelogram may make such a diagnosis possible by exclusion.

General Principles of Management

A patient with an established paraplegia or with premonitory or early symptoms and signs which might suggest cord compression requires a careful neurological and general physical examination. Three basic questions then require an answer: (1) Is the paralysis due to an intrinsic myelopathy or neuropathy (e.g., transverse myelitis or Guillain-Barré syndrome) or to compression of the cord? (2) If due to compression what segmental level is involved? and (3) What is the probable pathology?

If the clinical evidence is in favour of spinal cord compression then it remains to be decided if the lesion is intramedullary, extramedullary, or extradural in type.

The diagnosis cannot be exactly established without recourse to laboratory or radiological investigations. These include a red and white cell blood count (with indices and differential count if they are abnormal), an E.S.R., and a blood W.R. Plain *x*-rays of the spinal column and of the chest (which may reveal relevant pulmonary or mediastinal abnormalities) are invariably required and are generally to be followed by lumbar puncture and myelography prior to operation. In certain instances the radio opaque contrast should be introduced by cisternal puncture to establish the upper level of the compression, for example in the case of an extradural abscess.

The urgency with which these and subsequent measures are carried out will depend upon the suspected nature of the lesion. An acute and rapidly advancing paraplegia or paraparesis of a few hours' or days' duration such as may be due to compression caused by a metastatic growth or extradural infection demands all possible expedition in investigation and treatment and must be regarded as a surgical emergency no less pressing than acute appendicitis or a perforated peptic ulcer. More slowly progressive symptoms accompanied by early signs, while requiring prompt diagnosis, do not demand the same urgency. Included in this group are such conditions as benign spinal cord tumours, cervical spondylosis, and intrinsic disorders of the cord.

If, however, despite a long history the paraplegia or quadriplegia has reached a stage of complete or nearly complete paralysis investigation and treatment must clearly be instituted with all speed.

X-ray Examination of the Spine

Plain radiographs of the spine may show: no bony lesion; or pathological changes which are unlikely to be of significance—osteophytes, narrowed disc spaces, or simple osteoporosis; or changes in the pedicles or intervertebral foramina—long-standing local pressure from benign tumours may cause thinning of a pedicle and, in the case of "dumb-bell" tumours, widening of a foramen; or gross changes, such as vertebral collapse, paravertebral masses, or disappearance of pedicles, laminae, or spinous processes from malignant or chronic inflammatory invasion.

Such changes must be interpreted in the light of the clinical history and the general physical and neurological examination.

Lumbar Puncture and Myelography

Having established that the presenting symptoms are, or may be, those of cord compression the diagnosis is confirmed and the site of compression precisely localized by the findings at lumbar puncture and subsequent myelography.

In the presence of severe cord compression removal of

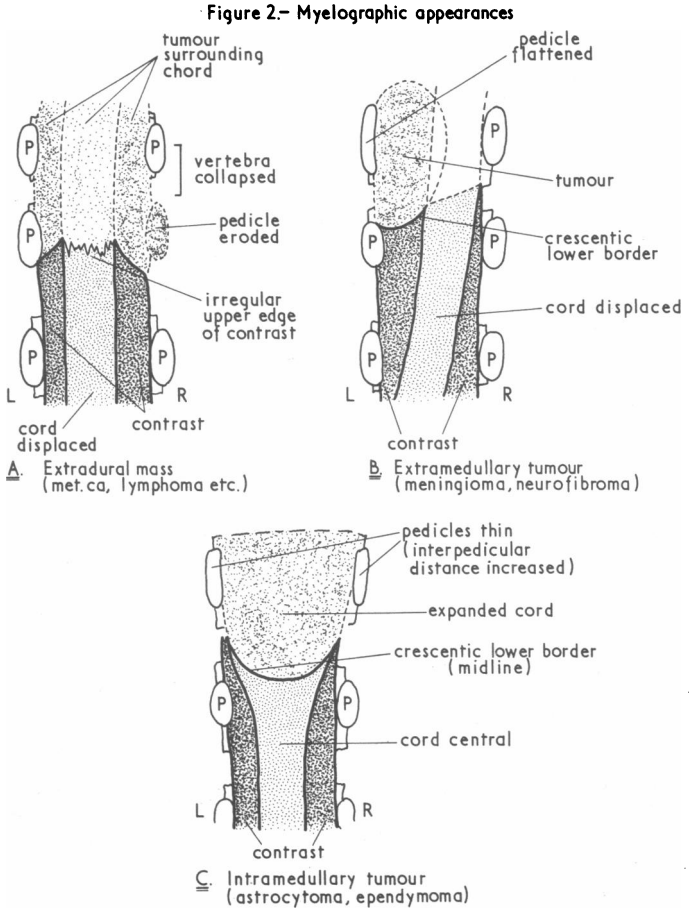
cerebrospinal fluid by lumbar puncture may result in worsening of the paralysis. Removal of the fluid from below the "block" may cause further impaction of the cord and tumour at the site of compression. Hence, it should be possible to carry out any definitive operative procedure as soon as possible after the lumbar puncture and myelography have been performed. When the cord compression is only slight or minimal—as in the case of cervical spondylosis or a benign tumour without a complete "block"—such deterioration is rare, and provided a careful watch is kept operative intervention may be deferred to the surgeon's and patient's convenience.

When there is no manometric evidence of compression a full examination of the fluid including the Lange curve and W.R. should be performed. The cerebrospinal fluid findings are then often suggestive or diagnostic of a non-compressive lesion. If compression is suspected or confirmed it is advisable to inject 6 to 9 ml. of contrast medium (Myodil) into the subarachnoid space before removing the needle because it may be difficult or impossible accurately to locate the subarachnoid space again for some days.

If it proves impossible to locate the subarachnoid space by lumbar puncture or when an extra-dural abscess or cauda equina tumour is suspected the Myodil should be introduced by means of a cisternal puncture. This is not a difficult procedure but requires caution and some experience.

The contrast material having been introduced, the patient is progressively tipped head downwards. The contrast is observed under a fluorescent screen (or preferably an image intensifier) as it flows from the lumbar region towards the head. From the appearances at the level of the block it is usually quite clear which type of obstruction is present. The characteristic appearances are depicted in the outline diagrams (Fig. 2). Myelography can be regarded as a "safe" procedure provided the necessity of urgent surgical intervention is observed in cases of acute or severe cord compression. Transient pyrexia occurs occasionally and rarely becomes severe or protracted. Aching in the legs and buttocks is common for a few days after the procedure. Untoward reactions leading to "arachnoiditis" are extremely rare.

Figure 2.- Myelographic appearances



Treatment

Throughout this article the urgency of establishing a diagnosis of spinal cord compression and carrying out definitive treatment has been repeatedly emphasized. The broad principles and indications for operative treatment have been discussed in the sections dealing with the various pathological processes. When postoperative radiotherapy is indicated it should be started forthwith—certainly by the time the wound is healed. There need be no fear of failure of the wound to heal provided there is no underlying haematoma or actual wound sepsis.

Stilboestrol therapy must be provided in cases of metastatic carcinoma of the prostate, and stilboestrol, testosterone, or prednisone may be required for certain cases of breast metastasis depending upon the particular circumstances of the patient. Chemotherapy is indicated for myelomatosis, but the solitary plasmocytoma of the spinal canal may best be treated by radiotherapy alone after macroscopically complete surgical removal. Cyclophosphamide should be administered in those cases of reticulosis when there is more than one focus.

General Care of the Paraplegic Patient

Nursing

Informed and active nursing care is essential for paraplegic patients. A two hourly turning regimen and rigorous attention to pressure areas at the same time are mandatory and may require additional staff. The skin of the whole body must be kept scrupulously clean by daily bed baths, particularly during the initial postoperative period. All pressure points must be suitably padded and protected.

The development of a pressure sore is heralded by the appearance of a rosy red patch which does not fade when the pressure is relieved. If vesiculation, superficial ulceration, or local gangrene of the affected area is to be avoided further pressure must be prevented forthwith by positioning the patient suitably. It is questionable whether the many remedies advocated for the treatment of incipient necrosis, ulceration, or gangrene have the least effect. The part should be kept clean and further pressure upon it proscribed. If local gangrene occurs the area should be excised when the sloughs appear and the cavity allowed to granulate. In a few cases, when the patient's condition merits it, pedicled grafts may be used to cover a large deficiency.

Care of Bladder and Bowel

In institutions devoted wholly to the care of paraplegic patients regular intermittent catheterization with strict aseptic precautions works well, and in these circumstances is probably ideal.

Where this is not feasible continuous bladder drainage using a No. 8 or 10 F. plastic Gibbon catheter (for males) or a No. 10 or 12 F. plastic Foley catheter (for females) introduced with full aseptic no touch technique and connected to a urine bag is as safe and least likely to be followed by urinary infection.

Whenever catheterization is required, whether intermittent or continuous, prophylactic oral sulphonamide therapy is essential accompanied by a large fluid intake. The urine should be examined initially and then at regular intervals for evidence of infection and the antibiotic or sulphonamide drug appropriate for the organism administered if infection occurs.

A patient with an acute paraplegia may develop gross distension of the bowel amounting to an ileus, presumably due to disturbance of its autonomic nerve supply. Gastric aspiration and intravenous fluids will then be necessary. Flatus may be relieved by the passage of a rectal tube. Constipation is a common feature of established paraplegia. If it does not respond to simple measures such as magnesia, liquid paraffin, and suppositories it may best be managed by a twice weekly colon washout or enema.

Relief of Pain, Painful Spasms, and Spasticity

Postoperative pain and pain due to local invasion of bone by malignant tumours is treated along the usual lines. After operations on the cervical spinal cord, however, it is wise to avoid morphine and its derivatives or substitutes because, immediately at least, depression of function of the phrenic outflow in the mid-cervical region may jeopardize respiration. Codeine phosphate by injection (25-60 mg.) can safely be used in these cases.

A particular problem in the management of severe spastic paraplegia is the occurrence of involuntary flexor or extensor spasms, which are often extremely painful. Much may be done to reduce their frequency and intensity by regular physiotherapy, by adequate rest and sedation, and by attention to any intercurrent infection in the bladder or elsewhere. There are no effective drugs available which will reduce these spasms nor the spasticity except the liberal use of morphine and its substitutes. In certain cases this may be desirable for short periods and may tide the patient over a bad patch, while in advanced malignant disease it may be entirely justifiable terminal medication. In other patients whose general health is good and life expectancy is not likely to be curtailed more definitive methods are available. These include obturator neurectomy to reduce adductor spasm and tenotomies of the hamstring muscles. These procedures, which do not add to the patients' burdens unduly, offer some useful relief both from spasms, spasticity, and pain. They do not disturb bladder function and obturator neurectomy makes micturition and defaecation easier. Another approach to the problem, particularly if voluntary control of the bladder is absent, is to paralyse appropriate roots of the cauda equina by instilling hypertonic saline (10%) or phenol (10-20%) in Myodil into the lumbar subarachnoid space, the latter under radiological control. This results in a flaccid paresis in the selected segments and thus may also affect the innervation of the bladder. In skilled hands this method is useful, but its reliability in producing the desired result cannot be guaranteed.

Rehabilitation

After an operation for spinal cord compression there must be an active regimen of physiotherapy. Regular, twice daily, supervised active movements for the normal limbs, thorax, and abdominal muscles are started at once, together with passive and where possible active movements of the paretic limbs.

Much will depend upon the aetiology of the compression, but in favourable cases re-education in walking should start in the gymnasium as soon as the return of power to the limbs permits. In some patients ataxia is a greater disability than weakness at this stage, and much patience is needed to allow time for progressive compensation to take place.

Patients who are unlikely to recover adequate function for walking but otherwise have a fair prognosis should be provided at an early stage with a suitable wheelchair which they can propel themselves. It cannot be emphasized too strongly how important it is that the morale of such patients should be maintained during the difficult period of readjustment. This is best done in the company of others similarly afflicted—the evidence for which is the success of the spinal injuries centres.

TODAY'S DRUGS

With the help of expert contributors we print in this section notes on drugs in current use.

Anthelmintics — I

Most of the worms that affect man live unobtrusively in the gut and do little to impair the health of their host. Symptoms and signs usually appear only with heavy infections. Rapid air-travel and the greater immigration into Europe from the tropics in recent years has resulted in helminthic infections, some of them long-standing and serious in their pathological effects, being encountered in Britain to an increasing extent.

While infections with particular types of worms are sometimes patchy in distribution and vary greatly in intensity in different localities, some sort of infection is almost always found in inhabitants of the developing countries with warm humid climates. In non-endemic areas it is justifiable to treat all infections however light, while in areas where re-infection is likely to occur only heavy or moderate infections are worth treating. The main aim of treatment should be to reduce the load of infection below the level of clinical significance. Complete parasitological cure is unnecessary. However, in patients who are severely ill from other causes such as protein-calorie malnutrition, marasmus, tuberculosis, or sickle cell anaemia worm infections, however light, should be treated even though reinfection is certain to occur.

Ascariasis

Ascaris lumbricoides has a world wide distribution, with the highest prevalence in Asia, Africa, and tropical America. During the migratory phase the larval stages may give rise to a pneumonitis with pulmonary infiltration and eosinophilia.

The piperazines are still among the most effective drugs for the treatment of ascariasis. Piperazine salts (the citrate, adipate, phosphate, or tartrate) can all be used, with little to choose between them. The dosage is 150 mg./kg. with a maximum single dose of 4 g. Neither fasting nor purgation is necessary. Side effects are rare; they include dizziness, paraesthesia, muscular incoordination, vomiting, and blurring of vision. These toxic symptoms are usually due to overdosage or occur when renal function is poor. The single oral dose of piperazine can be repeated on one or two occasions at intervals if necessary. Piperazine citrate or phosphate (Antepar) is a widely used preparation either as a syrup or in tablets. *Bephenium* hydroxynaphthoate (Alcopar) also causes a reduction in egg load in ascaris infection, and it is useful when combined treatment for ascaris worms and hookworm is required (see below).

Tetramisole has been used successfully in South America, South Africa, and Ceylon for the treatment of ascariasis. A single oral dose of 2·5-5·0 mg./kg. is claimed to produce cure rates comparable to those obtained with the piperazine compounds. More recently, satisfactory results have been reported